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ORIGINAL ARTICLES.

TEAR OF THE OPTIC NERVE BY A BULLET. HOLE AT THE MACULA.

By Ch. J. Kipp, M.D.,
NEWARK, N. J.

MICROSCOPICAL EXAMINATION OF THE EYE.

By Adolf Alt, M.D.

This left eye was removed January 16, 1908, from a boy 12 years of age, who had been shot four days previously. The missile had entered the frontal process of the malar bone, passed through the orbit and lodged in the cranial cavity.

The boy entered the Newark City Hospital shortly after he was shot. There was at that time great swelling of both lids, which were of a dark bluish color, and the eyeball was greatly protruded. It could not be moved in any way. The lower part of the ocular conjunctiva was ecchymosed. The cornea was clear; the pupil was widely dilated. The boy said he was blind in the eye.

I saw him for the first time on the day following the shooting. He was apparently in great pain and screamed when I attempted to separate the swollen lids. The eyeball was greatly protruded and could not be moved in any direction. The cornea was perfectly clear and *insensitive*. The pupil was widely di-

lated and did not contract on direct or indirect stimulation. The lens and vitreous body were perfectly clear. The disc could not be differentiated from the retina, which was a whitish color up to the visible periphery. The retinal vessels were exceedingly narrow, both arteries and veins, except a vein running from the macular region to the disc, which was of bright red color. At the macula was a deep red round spot in size about one-third of diameter of the disc. There was no perception of light.

Two days later I made another ophthalmoscopic examination and found the retina still more opaque, but the bloodvessels seemed to be all much fuller than on the first examination. There was no other change. As the boy developed symptoms of meningitis it was thought best to explore the orbit and if possible to drain the wound in the cranium. Accordingly on January 14th I detached the periosteum from the roof of the orbit and found that the bullet had splintered the roof of the orbit and had entered the cranial cavity. To secure better drainage I exenterated the orbit. No pus was found, but the outer wall of the orbit as well as the roof was broken into many fragments, which I removed. After the removal of the orbital tissue a large ragged opening could be seen in the roof of the orbit, from which brain substance was oozing. Shortly after this operation symptoms of abscess of the brain on *the opposite side* developed and the boy died on January 24, 1908, twelve days after the receipt of the injury. The exophthalmos was so great at the time I exenterated the orbit that the lids did not cover the eyeball and the cornea was exposed. Wet dressings kept on the eye did not prevent the development of ulceration in the upper and lower part of the cornea. The autopsy, made four hours after death, revealed a large abscess in the temporal lobe of the *right side* and a purulent meningitis of the convexity and base. The bullet was found in the abscess. An examination of the eyeball made immediately after its removal showed that the optic nerve and its sheaths had been torn by the bullet. The sheaths had been torn from the sclerotic and the sclerotic itself seemed to have been grazed by the bullet. The optic nerve was severed from the globe about 4 mm. back of its entrance. The eyeball was immediately put in a 4 per cent solution of formalin.

I may mention that we had radiographs made, one shortly after his entrance to the hospital and another after the exenteration of the orbit, but both failed to show the presence of the bullet.

REMARKS AND MICROSCOPICAL EXAMINATION.

Before giving a description of the interesting microscopical findings, I wish to report what was the macroscopical appearance of the eyeball.

In the first place, while part of the corneal epithelium was wanting, this was chiefly the case in the centre, the periphery all around being still covered with a band of epithelium. The pupil was wide, the lens opalescent.

When the eye was cut in two halves by an equatorial section, the retina almost to the very periphery appeared a grayish white. Here and there were a few small reddish brown spots, probably small hæmorrhages. The optic papilla showed no cup, but seemed flattened out so that it could hardly be differentiated from the surroundings. This agrees with what Dr. Kipp saw ophthalmoscopically. The retina surrounding the macula lutea was apparently very much swollen, so that the fovea centralis showed up much better than usual. In fact so beautiful appeared this picture, that before cutting up the eyeball for microscopical examination, I took occasion to show it to a number of medical societies. The retinal vessels were well filled and plainly visible, except in spots where the whitish exudation covered them.

The unusual picture of the fovea centralis made me particularly anxious to examine this part of the retina, and I was not very much astonished when the microscopic sections showed a rather large "hole in the macula."

This is the first hole in the macula I have ever encountered in all my microscopical work and I have never seen one ophthalmoscopically. Parsons in the last volume of his Pathology of the eye makes the remark (page 1163) that "no case has been seen ophthalmoscopically and examined microscopically—if my case of amaurotic family idiocy be excluded." I suppose his picture of the ophthalmoscopic appearance, Fig. 792, agrees well with what Dr. Kipp described in the history of the case under consideration as a "deep red round spot at the macula, about one-third the size of the disc diameter."

While holes at the macula seem to be caused without exception by injuries to the orbit or eyeball, during which a contusion of the eyeball takes place, it is the opinion of a number of observers that the rupture of the macular tissue is not due directly to the injury itself, but to the œdema which it produces.

Coats' opinion is evidently shared by Parsons, who sums it up thus: "Macular holes are produced by an œdema of the retina

at the posterior pole. The œdema may not be confined to the macular region, but the appearance of a hole will be produced, if there is a defect at least of the inner layers of the retina. Possibly for a completely typical picture without membranes or shreds a total defect of all the layers is necessary, and it has been proved that such a defect can arise from retinal œdema."

Parsons adds, "that the œdema may result from contusion (commotio retinæ), from toxins due to iridocyclitis, or from retinal vascular disease. Rupture of the retina *at the time of injury is not the cause of macular holes.*"

Whether this last statement is altogether correct in view of our case, remains doubtful. Dr. Kipp saw the patient the day following the injury, during which as he says the sclerotic at the posterior pole was apparently grazed by the bullet. On that day the red spot in place of the hole in the macula was plainly visible. Of course, there was also, extensive retinal œdema. Yet, I do not think we can with absolute certainty assume that the retina at this place was not torn at the very time of the injury.

Our case, according to Parson's statement, seems to be the first case in which a hole at the macula was seen ophthalmoscopically and examined miscroscopically.

In the June number of the *Klin. Monatsblätter für Augenheilkunde,* which I received a few days ago, Dr. C. Cohen describes a case of abscess in the choroid behind the macula lutea after a perforating guncap injury. In this case a rupture of the fovea centralis was directly due to the rupture of a localized choroidal abscess and therefore the case is not exactly of the same kind which we are here considering, while the histological picture of the hole is, of course, different chiefly only on account of the presence of pus.

It seems the cases to which the name of hole at the macula has been thus far applied were cases of a retinal defect not due to a choroidal affection, but rather with the absence of the latter.

The histological conditions of our eyeball are the following:

In all sections the corneal epithelium is wanting, except at the periphery. Bowman's membrane is intact, the clinically visible ulceration, therefore, was only a superficial one. However, the anterior two-thirds of the corneal lamellæ are throughout infiltrated with round cells. This round cell infiltration assumes such a character in the lower periphery of the cornea that here it forms an abscess. Descemet's endothelium seems unaltered

and its nuclei stain well. In the lower part particularly, but in the upper also, numerous polymorphonuclear leucocytes adhere to it. The meshes of the ligamentum pectinatum and the iris angle are choked with leucocytes.

The iris which has some astonishingly large crypts is moderately infiltrated, some smaller and larger accumulations of mononuclear and polymorphonuclear leucocytes lie on its anterior surface.

The ciliary body is enormously infiltrated with cells. The choroidal bloodvessels, especially near the posterior pole are very hyperæmic and distended. There is some cell infiltration in the tissue of the choroid.

Sections through the centre of the optic papilla and nerve show the central artery and vein well filled with blood and surrounded by a mantle of infiltration, the connective tissue bands in the nerve abound in round cells. The papilla, although swollen and pressed forward into the eyeball, as it is, is microscopically as flat on the surface as it appeared macroscopically. There is not a trace of a physiological cup. This is due to the swelling and œdema of its tissue. The retina on the macular side for some distance from the edge of the papilla consists apparently only of infiltrated nerve fibres the neuro-epithelial layers being absent. The first to appear outwardly from the papilla is the inner nuclear layers, and quite a distance further the outer nuclear layer makes its appearance; then come partly disintegrated rods and cones. This particular appearance might, perhaps, be due alone to the stretching by the œdema, and yet I cannot recollect having seen such an arrangement in other eyes with retinal œdema. I am inclined to think therefore that this condition in our eye is really due to a preexisting myopic conus.

In the macula itself the retinal tissue is especially swollen and the edges of the hole are bent somewhat away from the choroid into the vitreous chamber. There is some detritus containing some erythrocytes underlying the edges of the hole, and the pigment epithelial cells in the neighborhood are beginning to proliferate and to invade the outer layers of the retina.

The somewhat ragged edges of the hole do not show anything very particular, except that the cells nearest the hole accept the staining material but poorly. In a number of sections the inner limiting membrane of the retina is seen as a double contoured

wave line just as the lamina vitrea of the choroid appears when it is ruptured.

The size of the hole in our case is too large to be referred only to the fovea centralis; it evidently reaches somewhat beyond it into the macula lutea. Evidences of high grade œdema are visible everywhere in the retina, especially in the nerve fibre layers. The retinal bloodvessels are not only well filled with blood, but in a great many places they are so distended that they project beyond the level of the retina into the vitreous body.

The fact that the central retinal artery and vein as well as their retinal branches are so well filled with blood proves beyond a doubt that, even if the optic nerve was severed by the bullet, these blood vessels must have escaped a direct injury and the anæmia noticed in Dr. Kipp's first examination was probably due to compression by the undoubtedly great hæmorrhage into the orbital tissue.

CLINICAL THERAPEUTIC CONSIDERATIONS OF SYMPATHETIC OPHTHALMIA.*

By Dr. Ch. Abadie.
Translated by Adolf Alt, M.D.

At the last congress of ophthalmology some interesting discussions were had concerning the subject of sympathetic ophthalmia.

I asked several times for the floor to support new ideas and new therapeutic facts of which it seems useful to me that I make them known and that I resume this labor.

I put myself in the presence of an ocular injury of any kind, which has produced a sympathetic ophthalmia.

Of two things possible, one is that the affection is of such a nature that all hope of vision, all hope of saving even the shape of the eyeball is gone, the patient is definitely mutilated, disfigured. In this case enucleation is not a sacrifice, it is a deliverance. It is imperative.

On the contrary, if the wound is small, if there is still some vision, if the eyeball has retained its shape and its dimensions nearly normal, in such a case, I call it out loudly, we must not enucleate, at least not enucleate at once.

*Annales d'Oculistique, June, 1908.

We must search all the angles and points of the wound with the galvanocauter, wherever it is situated, no matter how much we destroy during this intervention.

Fromaget was right when he arose against the opinion of those who believed that such a cauterization with the galvanocauter could at any time provoke a sympathetic ophthalmia.

What we must say is, that sometimes in spite even of such a cauterization a sympathetic ophthalmia has supervened and this probably because some infectious focus had escaped the destruction by fire.

When by the complete cauterization the infectious focus has been destroyed, the loss of substance is covered by conjunctiva which is fastened over it by means of several sutures.

After this we await developments. Sometimes this is sufficient to abort the sympathetic ophthalmia. If it develops in spite of it we must employ mercurial inunctions and injections.

If the worst comes to the worst and the sympathetic disease is not aborted in spite of the cauterization, there is still time to enucleate.

In support of what I have just stated I can furnish the following observation:

My excellent friend, Dr. Dubarry, of Havre, sent me about a year ago a child, 8 years old, whose right eye had been injured by a mechanical toy.

Two weeks afterwards sympathetic ophthalmia made its appearance. Mercurial inunctions and subconjunctival sublimate injections were employed, but badly borne, and they did not cut short the progress of the disease.

The ciliary injection in both eyes increased, the media became more and more opaque and in proportion sight grew less. In this condition the patient was sent to me.

Without hesitating, on the day of arrival, I made in chloroform anæsthesia a deep cauterization of the wound, which was situated in front of the ciliary region. In doing this I felt certain that I should touch the equatorial edge of the crystalline lens and that a cataract would result therefrom.

This did not deter me from cauterizing deeply and penetrating into the ocular cavity. A drop of vitreous body presented in the wound when I withdrew the galvanocauter. I slid the conjunctiva over it and closed the wound.

At once the sympathetic ophthalmia was arrested and the patient got perfectly well with V=1 in one eye and V=2/3 in the

other. I had not touched the lens. If I had, I would have had a cataract which I would have operated on later.

Such cases in which both eyes have been saved prevent me from agreeing with Valude who advises preventive enucleation even when no sympathetic ophthalmia has made its appearance.

I agree with him in cases of considerable destruction with immediate loss of vision, but in the other cases, what criterion have we whether or not a late outbreak of sympathetic ophthalmia is to be suspected?

In the presence of an established sympathetic ophthalmia even, one must not act foolish, lose his cold blood and proceed to enucleate immediately when the injured eye is not irredeemably lost. Cauterize the wound deeply and vigorously with the galvanocauter. If the disease persists enucleate; if it still persists make mercurial injections and, if it still persists, make deep intraorbital injections.

This I have done to fight a case of sympathetic ophthalmia which developed in spite of enucleation and in spite of intravenous mercurial injections.

In a patient, whose history I have reported in detail (Congrès International de Médicine, Paris, 1900), injections of a few drops of a 1 in 1000 sublimate solution into the depth of the orbital tissue, brought about at once a very marked improvement in a sympathetic ophthalmia which had yielded to no previous treatment. But soon when the improvement thus obtained did not progress, it became necessary to inject 5 or 6 drops of a 1 in 100 solution in order to obtain a perfect cure. These injections were very painful and almost caused a collapse.

Since then other observations have been published and they are becoming more and more numerous in which a sympathetic ophthalmia, having resisted the enucleation, mercurial inunctions or injections, has yielded finally to intraorbital injections of a solution seldom surpassing 1 in 1000 in strength.

There are some cases of late sympathetic ophthalmia, whatever else may be said.

They are usually due to the fact that the wound resulting from the injury is but incompletely closed and not covered by a protecting epithelium.

In such cases we must resort to the same treatment. Here, too, we must cauterize with the galvanocauter, searching all the

corners and folds, and cover the wound with conjunctiva held in place for some days until it is firmly united with the freshened wound.

Finally, it may happen that, although a sympathetic ophthalmia has developed, the patient refuses the enucleation.

In such a case we must make intraocular injections of one or two drops of a 1 to 1000 sublimate solution into the injured eye, as I have recommended many years ago.

Terson, the father, has reported two cases from his practice, in which patients suffering from sympathetic ophthalmia refused the enucleation and in which he injected a drop of 1 in 1000 sublimate solution into the injured and lost eyeball. In both these cases the sympathetic symptoms were stopped and although the patients were kept under observation for a number of years, no relapses occurred.

ON REGIONAL ANÆSTHESIA IN THE ORBIT.*

By Dr. A. Loewenstein.

(From Prof. Dr. A. Elschnig's Clinic, Prag.)

Translated by Adolf Alt, M.D.

For years numerous trials have been made to replace the general anæsthesia and its dangers by local anæsthesia in cases of enucleation of the eyeball.

Snellen[1] reduced the sensibility of a very painful eyeball by injecting cocain into the vitreous body by means of a Pravatz syringe. Schleich[2] reported that he had performed enucleation by injecting his cocain solutions (0.2%, 0.1% and 0.01%), but gave no detailed rules. Later on Weiss[3] tried Schleich's method in five cases, in which he made use of Schleich's general rules for local anæsthesia. Anæsthesia follows only where the anæsthesizing fluid penetrates; cocainization of the conjunctiva (2%), subconjunctival injection of Schleich's solution, and then on both sides greatest possible infiltration. Yet the operation was not totally free from pain, especially in the cases in which a chronic inflammation of the eyeball had existed.

A similar communication was made by Hackenbruch,[4] who succeeded in producing perfect anæsthesia with Schleich's infiltration.

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*Klinische Monatsblätter für Augenheilkunde, June, 1908.

Haab[5] injected his weak cocain solution at first at the insertions of the four recti muscles and then, after the cutting of the tendons, injected a considerable quantity of a cocain eucain mixture into the neighborhood of the posterior pole of the eyeball by means of a curved canula. In appropriate cases he had a good local anæsthesia after five minutes. He restricted this method, however, to cases in which no obliterations of Tenon's capsule existed, that is noninflammatory ones.

From a general surgical standpoint H. Braun[6] recommends Haab's method in two times, that is, to first infiltrate with 10 cubic centimetres of his solution from four points all the soft parts between eyeball and periosteum, to detach the tendons of the four recti muscles and then again to inject 10 cubic centimetres at four points behind the equator of the eyeball as far back as possible with a slightly curved needle around the optic nerve.

Otto Meyer[7] makes the tenotomy of the four recti muscles after having circumcised the conjunctiva after ordinary cocain instillations; after this he proceeds according to Schleich's infiltration method. He, also, mentions that Herrnheiser had made at the Prag eye clinic in 1887 seven enucleations under subconjunctival and retrobulbar anæsthesia.

A little while ago A. Siegrist[8] published his method which is no longer, as are the others, based on Schleich's infiltration. He first cocainizes superficially, then with a curved canula, its concavity toward the eyeball, he injects between eyeball and orbital wall on the four sides behind the eyeball towards the entrance of the ciliary nerves each time 0.75 cubic centimetres of a 2% novocain solution with a small admixture of adrenalin. He states that even with inflamed eyeballs he had good results with this method. He thinks that the ciliary nerves at their entrance into the eyeball are paralyzed by the novocain.

My chief, Professor Elschnig, like many other operators, has since 1892,[9] enucleated eyes under local anæsthesia with a 2% cocain solution. While successful with uninflamed eyeballs, this method was totally unsuccessful with inflamed and painful ones.

Thus, Professor Elschnig, was driven to look for another method which would, also, anæsthetize perfectly a painful eyeball and permit of enucleation and exenteration under local anæsthesia. He, therefore, contemplated a regional anæsthesia from the ganglion ciliare.

While previous methods, even Siegrist's injections on four sides, were in principle based upon the infiltration with the anæsthetizing fluid of the tissues about to be cut, our method may be called a conduction anæsthesia.

As Feinberg and others have first shown in animals, and Corning and Goldscheider in man, cocain has the faculty of destroying both the motor and sensory nerve conduction. The surgeons have for some time been making use of the endoneural injection (Crile) with the same success as with perineural injections (Matas). The latter is the same as our method.

The nerves which render the eyeball sensitive are the long ciliary nerves coming from the naso-ciliary nerve and the short ones coming from the ciliary ganglion.

The long ciliary nerves arise from the naso-ciliary nerve very shortly after it has sent the long sensitive root to the ciliary ganglion.

Thus we see, that all sensitive nerves for the eyeball lie close together, in the neighborhood of the ciliary ganglion. An injection directed against the ciliary ganglion affects all sensitive nerves of the eyeball.

The task was to find a simple and certain way to find the ciliary ganglion from the surface. At the instance of Prof. Elschnig I began the researches in the anatomical institute of the German University. Professor Fick, whom I want to thank here, very kindly put at my disposal the specimens of the institute. On the five ganglion ciliare specimens I measured its distances from a fixed point. For this I chose the middle of the lateral orbital margin. The average distance was 4½ centimetres. I then entered the orbits of corpses laterally from the eyeball with a long enough canula on the syringe (the ciliary ganglion lies laterally of the optic nerve), always followed the lateral orbital wall until the point of the syringe had entered 4½ cm. as measured from the middle of the lateral orbital wall. Leaving the canula in this position, I chiselled off the orbital roof and dissected the ciliary ganglion. I found that the point was exactly in front of the ganglion. When examining the route the canula had taken, I found the external rectus perforated, but the optic nerve and the larger bloodvessels uninjured. A repetition of this experiment resulted in the same way.

Based on these experiments Professor Elschnig made a series of operations under ganglion anæsthesia, which he bade me report.

The procedure was as follows: Usually the conjunctiva was cocainized, then a Pravatz syringe filled with a 1% cocain solution was armed with a strong needle 5 centimetres in length. The lateral lid commissure being drawn temporarily the needle was entered through the conjunctiva close to the lateral orbital wall and a little below its middle, corresponding with the lower edge of the external rectus. Thus the perforation of this muscle and the pain caused by it in inflamed eyeballs is avoided.

The needle glides along the lateral orbital wall; lastly it is pressed somewhat inwards so that about ½ centimetre of the canula still remains outside the conjunctiva and the whole syringe full is slowly injected.

It is well to satisfy oneself by slight movements that the point is not in the optic nerve or in one of the larger bloodvessels. My experiments on the dead body showed me how easily the needle penetrates the optic nerve if one tries to reach the ganglion without such precaution or when forcing the needle in too deeply. I could inject a whole cubic centimetre of red ink into the optic nerve without getting a drop into the orbital tissue.

Almost immediately after the injection, at the longest after 2 minutes, in every case the eyeball is anæsthetized. Pressure on the cornea or on the ciliary region is not felt at all or simply as a painless touch, even in eyes which were previously enormously painful from a cystitis or beginning panophthalmitis. Exenteration could easily be done in such cases without pain. In by far the most of the cases the remainder of the orbital contents was also anæsthetic, so that dragging on the eyemuscles during an enucleation was perfectly painless; certainly neither the cutting of the optic nerve nor of the ciliary nerves was felt. However, in some cases, especially in eyes which had suffered from a chronic inflammation, the cutting of the conjunctiva was still felt as pain, when the formerly painful eyeball was perfectly anæsthetized. Yet an additional subconjunctival injection of cocain brought about the desired result, so that now we always add one in such cases. Probably in these cases a new formation of nerve tissue has taken place by which, as is the case with new formed bloodvessels, new and alien nerve territories are influenced.

We now employ a 1% cocain solution with a few drops of adrenalin. Since we hardly ever use more than 1½ or 2 syringefuls, that means 1.5 to 2 cubic centimetres or 0.015 to 0.02 g. cocain, we are still far from Woelfler's maximal dosis of 0.05 g. Pro-

fessor Elschnnig does not fear intoxication with cocain as does Siegrist. Like all others who use the 1% solution (Reclus, Schleich, Oberst, etc.) we have never seen the least disagreeable effect from our method of using cocain.

We recommend now the following procedure to find the ganglion, and the nerve trunks at the apex of the orbit:

The needle is entered as described and pushed forward with the necessary precautions, then half a syringeful injected. While the needle is left in the orbit we see by pressing on the eyeball with forceps whether it is anæsthetic. If not, we withdraw the needle somewhat and again push it forward in a different direction. The correct position of the needle will be shown (before injection) by pain being caused when moving it.

When the eyeball does no longer feel pain from pressure we inject in the inflammatory cases some cocain subconjunctivally, about 15 mm., from and all around the limbus; in most cases the conjunctiva is anæsthetic.

Finally a rather hard pressure should be employed to make sure that the eyeball is perfectly without sensation.

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We have done 26 operations under ganglion anæsthesia, 18 enucleations, 7 exenterations and 1 cyclodialysis in a painful amaurotic eye. Thirteen of these cases had been painful eyes in consequence of chronic inflammation.

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The anæsthesia is confined to the orbital tissues exclusively. The other branches of the trigeminal remain in function, as sensitive as the mucous membrane of the nose and mouth. When the needle had almost reached the ganglion several persons felt a sudden transitory pain in the nose by projection through the naso-ciliary nerve.

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We must mention that most of the patients, as before other operations, received half an hour before a subcutaneous injection of 0.01 g. morphium, which does not influence the pain, but has a beneficial influence upon the psychical tenor of the patient during the operation.

During this time we have never seen any disagreeable symptoms from our method of using the cocain; after the operations the patients felt perfectly well.

The anæsthesia from the ciliary ganglion or at least from the apex of the orbit, according to our experience, is practicable and

equally successful in cases of enucleation and evisceration of the eyeball, not only in noninflammatory, but, also, with painful eyes. Its application is, technically speaking, easy, rapid and without disagreeable consequences.

Whether it is indicated also in other painful eyeball operations, as iridectomy and cyclodialysis, will depend on further anatomical studies and experiments on animals. From these we, further, expect to learn whether injection into the region of the ciliary ganglion or the naso-ciliary nerve may be employed for therapeutic purposes, as similar injections have in recent times been employed successfully in other sensory nerves.

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MEDICAL SOCIETIES.

THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

Thursday, June 11th, 1908.

The President, Mr. Marcus Gunn, in the chair.

PAPERS.

An Epidemic of Contagious Conjunctivitis due to the Pneumococcus.—Mr. P. H. Adams.

This paper dealt with an outbreak of conjunctivitis which occurred in a school containing 37 boys, 20 of whom became infected. The features of this particular type of conjunctivitis were some aching in the eyes at the commencement, followed by sticking of the lids two days later, slight redness of the conjunctiva, and lacrimation. There was only a small amount of discharge. The affection appears usually in the spring, is not acute, and is very contagious. It is chiefly confined to children, and lasts from a week to a month. Smears were taken in almost every case, and gram-positive diplococci were found, some of which were definitely encapsuled. A growth on blood agar revealed typical pneumococcus.

In answer to some questions afterwards Mr. Adams said that inoculation experiments were not undertaken.

Lower Corneal Plaques.—Col. H. Herbert.

These are slightly raised greyish patches, with rough surfaces and feebly vascular, found occasionally in patients in India close to the lower margin of the cornea. General pannus was found associated in only a few instances. Apart from the curious shape of some of the patches the chief point of interest is the ætiology of the condition. The patches appear usually to be due to abnormal exposure of this strip of cornea, together with chronic conjunctivitis, trachomatous or simple. But the cause of the defective closure of the lids was often obscure, and in some cases

there was absolutely no evidence obtainable of defective closure. And the condition was not found with paralysis of the orbicularis muscle.

A Note upon Secondary (Parenchymatous) Xerosis.—Col. H. Herbert.

Attention was drawn to the influence of general pannus of both eyes in the production of xerosis secondary to old trachoma. The pannus renders the corneal surface somewhat insensitive; thence the blinking movements of the lids become infrequent and incomplete, and exposure of the cornea thus brought about adds to the loss of sensibility. In consequence of this the reflex action of the lids in some cases remains almost in abeyance. Thus there is a striking want of relationship between the xerosis and the cicatricial degeneration of the conjunctiva, to which the xerosis is ordinarily entirely attributed. In some cases of very marked retraction of the conjunctival fornices the cornea remains perfectly clear and moist, whereas in other cases with much less advanced cicatricial contraction the secondary xerosis is well marked.

Among other factors in the development of cicatricial xerosis should be noticed the reduced transudation of fluid from the general palpebral conjunctiva. If the upper lid be everted in an ordinary case of acute conjunctivitis, a very considerable flow of fluid from the exposed conjunctiva is readily demonstrable. A similar outflow is not obtainable from a cirrhosed (diffusely cicatrized) conjunctiva, however much conjunctivitis be present. In these shrunken conjunctivæ there is a dense and thick basement membrane.

Senile Cataract in Husband and Wife—Condition of the Lenses in Children and Grandchildren.—Mr. E. Nettleship.

The husband and wife were both operated on for cataract, and of their 10 children 3 females had mature cataract, while 3 males and 1 female had incipient cataract; only the eldest and the youngest had escaped; and one was dead. Of the grandchildren 5 males had incipient cataract, though the changes were very scanty.

Mr. Nettleship discussed the relation of this family tree to the Mendelian theory. If the liability to cataract behave as a "dominant" several of the children ought to have shown it. In order

to explain in simple Mendelian terms the incidence of cataract in the third generation, it must be assumed that the parents were "impure or incomplete dominants," each carrying the normal character obscured or hidden, but not destroyed, by the cataractous character, and this required that one of each of their parents should also have had cataract. As, however, it happened that the parents all died before the age at which it would develop the point can neither be proved or disproved, but the assumption is a fair one. If the parents be "impure dominants" their offspring should, on the Mendelian theory, consist of dominants and recessives in the proportion of 3 of the former to one of the latter. The proportion of children in this genealogy who developed cataract to those who were normal was as three to one.

Lamellar Cataract, Coppock or Discoid Cataract, and Retinitis Pigmentosa, affecting different members of the same Pedigree.—Mr. E. Nettleship.

This genealogical tree related to 275 persons, of whom 110 were males, 115 females, while of 50 the sex was not recorded. All are descended from 2 brothers, and the chart is divisible into 2 parts, the larger (about 180), containing numerous cases of lamellar and Coppock cataract, and the smaller 32 cases of cataract and 15 of retinitis pigmentosa out of a total of 150. The presence of these two conditions associated in the same genealogy is doubtless due to each disease having been introduced by different ancestors from independent sources; and the fact that the retinitis pigmentosa is found only in the descendants of one brother inclines one to assume that the taint has been introduced through the wife. The pedigree also shows that Coppock cataract can develop in a child from a parent with lamellar cataract.

A Color-blind Family.—Mr. E. Nettleship.

This chart dealt with an attempt to examine the color vision, by the ordinary wool tests, of all the available members of a rather extensive pedigree of educated people. The three current generations contained about 110 persons; and the genealogy showed, so far as it went, the ordinary descent of this defect, viz.: all the offspring of the color-blind ancestor escaped, but in the next generation a certain number of the sons of some of the daughters were color-blind.

Accompanying these three papers was an exhaustive appendix, with details of every case examined.

CARD SPECIMENS.

Sky-blue Sclerosis.—Mr. G. Winfield Roll.

This was the case of a young woman whose sclerotics were of a uniform pale blue color, a condition which had existed since birth. In all other respects the eyes were normal, and the vision with correction 6/6.

Double Optic Neuritis of doubtful Syphilitic Origin.—Mr. G. Winfield Roll.

A male patient, aged 29, showed optic neuritis of both eyes, the right disc being more swollen than the left, with a good deal of œdema and some exudation in other parts of the fundus. There was a history of the primary sore in 1902 with slight eruption later, and ulceration of mouth 4 years afterwards. The right eye began to fail early in 1907, and the left in August of the same year. Mercurial treatment was adopted, but the vision gradually deteriorated until at the present time there was no P.L. in either eye.

A curious Congenital Anomaly of the Iris.—Mr. S. Stephenson.

In a male baby aged 15 months, brought for advice in May, 1908, was found in the right eye a small oval pupil situated in the upper nasal quadrant, and on an anterior plane to this and partly covering it, was a curved piece of tissue of a light-blue color, occupying the upper and inner part of the anterior chamber. At the extreme periphery in one part of this was a narrow zone of what appeared to be iris. No history could be elicited to account for this abnormality.

Connective Tissue obscuring the Left Disc.—Mr. L. Paton.

A male patient, aged 58, came with the history that the sight in the left eye had been bad for years; it was found to be 6/60. A white structureless growth was seen to obscure almost entirely the left disc, the retinal vessels disappearing under the edge of it. To the nasal side of the disc was a patch of choroidal atrophy with some pigmentary degeneration of the retina over it. Some white glistening spots appeared on the temporal side, with horizontal striation of the retina beyond. The right fundus exhibited arterial thickening.

Thiersch Graft of the Orbit.—Mr. A. C. Hudson.

This was the case of a young man in whom exenteration of the right orbit had been undertaken for sarcoma in November, 1907. A month later, when the orbital surface had become covered with granulations, these were lightly rubbed over with gauze, and the whole cavity was lined with a single Thiersch graft from the arm. A protective gauze dressing was applied and firmly packed with cotton wool. It was dressed for the first time 6 days later, when the graft had united thoroughly, all the raw surface being completely covered.

A peculiar form of Retinal Disease.—Mr. A. Stanford Morton.

Henry H., aged 26, came for treatment with history of failure of vision in the right eye for one month, supposed to have come on suddenly; but this eye had always been divergent. The vision was hand movements at 3 in. and projection was bad except in the lower part of the field. The media were clear.

A colored drawing of the fundus was exhibited which showed enormous masses of exudation in all parts of the fundus except upwards and outwards; the macula also was fairly free. The exudate was most dense in the neighborhood of the main vessels which were pushed up by it. The disc was hazy but appeared otherwise normal. In the peripheral parts of the fundus the vessels showed many varieties of shape, size, and tortuosity, while here and there some hæmorrhages were visible; there was a certain amount of pigment scattered about, and in the upper part a mass of fibrous tissue projected forwards into the vitreous.

A month and a half later the eye was removed for secondary glaucoma.

The pathological report, by Mr. G. Coats, stated that the white exudate proved to be mostly due to swollen leucocytes in the subretinal space. The retina itself was thickened, degenerated, and widely detached. The mass of fibrous tissue was situated between the retina and the choroid, but more intimately connected with the former, and it contained blood and cholesterin crystals. The bloodvessels, which in some places were thrombosed and enormously dilated, showed a peculiar hyaline degeneration of their walls.

MALCOLM L. HEPBURN.

REVIEWS.

THE PATHOLOGY OF THE EYE. By J. Herbert Parsons. Volume IV. General Pathology, Part II. New York: G. P. Putnam's Sons, 1908. London: Henry Frowde and Hodder & Stoughton.

With this volume the monumental work of Parsons comes to conclusion. As we have stated at the appearance of the previous volumes, this is a work of superior merit and author and publishers are to be congratulated at the result of their work.

The volume of information contained in the pages of this book is too great to be analyzed by the reviewer. Surely every English speaking oculist should have it on his shelves for reference. I am sure no one working in the field of eye pathology can afford to be without it. ALT.

ICONOGRAPHIE STEREOSCOPIQUE OCULAIRE. Par le Dr. A. Monthus. 25 plates, with explanatory text. Masson & Co., Paris, 1908. Price 5 francs.

This collection of stereoscopic photographs concerning the anatomy of the orbit and adjacent cavities and a number of pathological conditions, and finally a number of operations on the lids and lacrimal apparatus, is very pleasing and will undoubtedly help the student in reading to get a better understanding. The selection of the subjects in this collection appears somewhat peculiar, as they follow no logical line and are but loosely connected. Maybe they form only a part of a larger atlas in the future. ALT.

CONTRIBUTIONS TO THE SCIENCE OF MEDICINE AND SURGERY BY THE FACULTY, in celebration of the 25th anniversary of the founding of the New York Post-Graduate Medical School and Hospital. 1908.

This beautiful and truly representative volume contains 48 articles by different members of the faculty in all its branches. Among these we find three which more particularly concern the ophthalmologist.

The book, which is in beautiful large type and furnished with excellent illustrations, is furthermore graced by an excellent etching of the late Dr. D. B. St. John Roosa, and his biography, written by Dr. Ch. L. Dana, a fit tribute to the memory of the founder and president of this great medical school to whom it was not given to live and to receive this book which was originally intended to be a testimonial to him. ALT.

ABSTRACTS FROM MEDICAL LITERATURE.

By W. A. Shoemaker, M.D.,
St. Louis, Mo.

MYOPIA ASSOCIATED WITH OPACITIES OF THE CORNEA.

Walter L. Pyle (*Jour. A. M. A.*, July 20, 1907) suggests the following conclusions from his study of this subject:

1. Central corneal opacities are a much more frequent cause of myopia than would be inferred from an examination of general ophthalmic literature, particularly the English and American text-books and periodicals. According to recent reliable Continental statistics, in at least one-third of the cases in which they are present, central corneal opacities produce myopia, although this average is likely higher than that found in American ophthalmic practice.

2. The myopia is axial and not dependent on alteration of the corneal curve. Ophthalmometric examinations do not show any significant differences between the corneal curvature of myopic eyes with corneal opacities and similar eyes in which the refraction is emmetropic or hypertropic.

3. The mechanism of the production of the myopia is analogous to that of the common form following astigmatism, anisometropia, etc., with the added predisposing factor of weakened ocular tunics by previous inflammation.

4. The association of disturbance of the chorioid and retina of the posterior segment of the eyeball with inflammations of the anterior membranes is an important element in further reducing visual acuity in these cases. For a similar reason extensive fundus lesions are common, and the myopia is more likely to become progressive than ordinary.

5. Either unilateral or bilateral opacities may produce myopia, and the myopia produced may be unilateral or bilateral.

6. In the cases of unilateral opacity the myopia may be single or double. If in such cases the myopia is unilateral it may appear either on the sound or the affected side, but no definite rule can be established in this connection. However, it is likely that when one eye is used almost exclusively for close vision it will become myopic first or to a greater extent than its fellow.

7. In cases of bilateral corneal opacities the myopia is gen-

erally bilateral. When there is a marked difference in the amount of defect in the two eyes the greatest myopia is often found in the eye with the least corrected visual acuity. When the myopia is unilateral it often occurs in the eye with the least opacity.

8. The cases of unilateral myopia are strongly suggestive of the importance of ciliary strain rather than excessive convergence in myopogenesis.

9. In view of the strong possibility of resultant myopia, all cases of corneal disease, particularly the phlyctenular affections of childhood, should receive prompt and continuous treatment, and prophylactic measures should be rigidly followed to prevent recurrences. After the subsidence of the inflammation energetic treatment should be adopted to clear up the opacities.

10. Of greatest importance in the prevention of subsequent myopia is careful and repeated refraction of both eyes under artificial cycloplegia, employing both objective and subjective methods, and the constant use of correcting lenses.

11. All errors in ocular, personal and domestic hygiene that encourage the development of myopia should be corrected, and the patient should be kept in the best physical condition and most salutary environment.

A METHOD OF PERFORMING TENOTOMY WHICH ENABLES THE OPERATOR TO LIMIT THE EFFECT AS REQUIRED.

Frank C. Todd (*Jour A. M. A.*, July 13, 1907) describes his method of performing tenotomy for the relief of heterophoria or strabismus. Instead of doing a complete tenotomy by which method the effect cannot be well regulated, and the tendon may be reattached in such a position that muscle imbalance not previously existing may be developed; or doing a partial tenotomy as recommended and performed by Stevens, which often is of little value, he exposes the tendon and having passed a tenotomy hook under it makes an incision in one edge of the tendon close to its attachment to the globe on the corneal side of the hook. On the opposite edge of the tendon and at about two-thirds of its width from the first incision he makes a second incision which will be on the muscle side of the hook.

The effect desired can be regulated by the length of the incisions, and measurements can be taken to enable the operator to get precisely the amount he wishes.

The author cites the following facts concerning the operation:

1. This method may be practiced in cases of heterophoria; the muscle balance measured by the Maddox rod or the prism test at any stage of the operation and the effect increased a little at a time (as low as one-half degree if desired) until the proper effect is secured.

2. In those cases of strabismus in which it is impossible to secure single binocular vision, measurements may be made by the parallax test (of Duane) or by the cover test and the effect regulated at will.

3. This operation may be performed in conjunction with advancement operation on the opposite tendon.

4. The possibility of the wrong insertion of the muscle is prevented, as the insertion is not interfered with, the muscle simply being lengthened.

5. The operation may be painlessly done under local anæsthethesia and the eye left unbandaged.

THE OPTIC NERVE CHANGES IN MULTIPLE SCLEROSIS.

Ward A. Holden (*Jour. A. M. A.*, July 11) discusses:

a. The pathologic changes in the optic nerves in multiple sclerosis.

b. The types of fields of vision, and the corresponding ophthalmoscopic changes in the optic discs.

c. Retrobulbar neuritis that is probably an early symptom of multiple sclerosis.

d. The causes of retrobulbar neuritis other than multiple sclerosis.

He recapitulates thus: Disturbances of vision are found in about half of the cases of multiple sclerosis. Usually the dimness of vision is noticed early in the course of the disease, and it may come on long before any other symptoms have attracted attention.

More than half of the patients with multiple sclerosis who complain of failing sight have central scotoma in the field of vision, and the ophthalmological diagnosis of their condition is retrobulbar neuritis. Toxic cases of retrobulbar neuritis, due to poisoning with tobacco, alcohol, lead, and the toxins arising in pregnancy are readily recognized and diagnosed as toxic. The causation of non-toxic retrobulbar neuritis, however, has often

been obscure. We have learned recently that many of these cases are due to sphenoidal and particularly to ethmoidal disease, which involves the optic nerve in the optic foramen.

In my opinion, we shall soon come to the belief that a non-toxic retrobulbar neuritis, if not due to a sinusitis or directly to syphilis, diabetes, a neoplasm, or trauma is, as a rule, a manifestation of multiple sclerosis, although no other symptoms of the disease may be present.

DEEP INTRAOCULAR LESIONS OF INFANTILE INHERITED SYPHILIS AND TARDY INHERITED SYPHILIS, FROM THE STANDPOINT OF GENERAL PATHOLOGY, PROGNOSIS AND TREATMENT.

C. S. Bull (*Medical Record,* April 4th) states that in his experience prenatal disease of the deep structures of the eye is not as infrequent as is usually supposed, and urges that an earlier and closer examination be made of the eyes of children whose parents are known or supposed to have syphilis. Such diseases as iritis, cyclitis, choroiditis, cataract, exudates in the vitreous, and less frequently, retinitis, optic neuritis, and optic atrophy have been found in infants several hours after birth, and he believes that disease of the uveal tract is more common in infants than is keratitis. He describes five cases of actual congenital syphilitic disease of the eye. The diagnosis is more difficult in tardy inherited syphilis. All obscure conditions must not be attributed to inherited syphilis, but a close examination should be made of the patient and also of the family, if need be. Evidences of syphilis may appear in the eye ground as gross lesions and also as rudimentary signs of previous lesions. Such functional disorders as amblyopia, night blindness, nystagmus and apparent myopia may occur in tardy inherited syphilis. Specific treatment is indicated in all cases where lesions of possible syphilitic origin exist, particularly if any undoubted stigmata are present. A proper dietary is very important for infants. If other children in the family show evidences of inherited syphilis, the parents should also be placed on specific treatment.

THE TREATMENT OF INTERSTITIAL KERATITIS.

H. H. Martin (*Jr. A. M. A.*, April 11) believes that the treatment of interstitial keratitis resolves itself into the treatment of congenital syphilis and that the best treatment of this is mercury, preferably by inunction. In children under ten years of age he begins with ten grains of a carefully prepared blue ointment which is increased to fifteen or twenty grains. In children over ten years of age or in adults ten to thirty grains constitutes the first dose, increased to twenty or thirty grains in children and to sixty in adults. This is used every other night after a hot soap and water bath, regulating the size of the dose by the effect produced and aiming to keep the patient on the verge of ptyalism. He thinks that iodids, during the inflammatory stage, are useless, if not harmful. His experience has led him to think that local measures are of no value during the stages of infiltration and vascularization, except to relieve symptoms and complications.

To assist resolution yellow oxide of mercury ointment, calomel dusted in the conjunctival sac, or subconjunctival injections of normal salt solution may be used, but are generally not needed. The patient should be kept under observation for six months after recovery and should take moderate sized doses of mercury by the mouth at intervals, and iodid of iron, with plenty of fresh air and generous diet of good food.

OCULAR ROTATIONS IN PARESIS.

Francis Valk (*Jr. A. M. A.*, April 11) summarizes his article as follows:

All tests for paresis must be made with the look in complete infinity and with full acuity of vision.

We must have a standard of the field of version for comparison of any deficiency of rotation.

The straight muscles are capable of moving the eyes in any part of the field of vision and from one part of the field to another; in other words, in no part of the field will the straight muscles fail to act according to their usual function.

The obliques take no part or action in the rotation of the eyes in the field of version; in other words, the oblique muscles do not elevate or depress the visual line.

A distinct line must be drawn between paresis and heterophoria as shown by the diplopia; in the first by diplopia in in-

finity and in the second by the possible diplopia with reduced vision.

In consideration and diagnosis of paresis we may exclude the condition of crossed or homonymous images and also the tipping, except in a pathologic condition of the obliques.

The individual muscles of the eyes are not endowed with two distinct functions, as they have but one nerve supply, except possibly in the action of convergence and inward rotation due to two distinct nerve centers.

The points of action of the straight muscles of the eyes are the insertions and the sheaths of the tendons as they are situated in the orbit just behind the globe.

MAJOR SMITH'S OPERATION FOR EXTRACTION OF CATARACT IN THE CAPSULE.

J. Rutter Williamson (*Ophthalmoscope,* Oct., 1907), after reviewing briefly the technique of Smith's operation and some of the conclusions Smith has reached after performing it over 14,000 times, summarizes the advantages and disadvantages of the operation as follows:

"1. No after-cataract to be dealt with, causing the patient disappointment that the one operation was not enough, and necessitating further absence from home and work, as well as the risks attendant upon the treatment of such after-cataract. We must all know how often a patient refuses to have anything further done, and then his poor visual acuity discredits the surgeon and hospital where he was first treated.

"2. Lessened risk of iritis from there being no lens-matter left behind.

"3. No need of introducing instruments, such as iris forceps or irrigator. This minimum of instrumentation must also mean a minimum of risk of inflammatory and infective processes.

"4. The ease with which immature cataracts can be removed at one sitting.

"5. The shortened period of stay in hospital, due to the absence of complications. The rareness with which atropin is used is striking, and is, in a large hospital, an appreciable saving of the assistants' time.

"6. One might also add that the avoidance of a conjunctival flap in Smith's operation is certainly convenient in keeping the

field clear from blood, and it does not seem to make healing the least bit slower. I rather doubted this at first, but after further experience I think that, as far as one can judge, the healing with the corneal flap seems to be every whit as rapid and as sound."

Against the operation, however, must be put:

"1. Greater skill necessary for its performance.

"2. An intelligent assistant requisite.

"3. A real, though not a vastly greater risk of vitreous escaping than in the usual capsule-laceration operation. This risk, like many others in surgery, becomes less as the operator becomes more expert. But, when every allowance is made, it does constitute one of the real dangers of the operation. As a rule, if Smith's directions are followed, the amount escaping is small.

"4. Possibly consecutive upon 3 is also a greater risk of remote retinal detachment.

"5. Another possible, though as far as I can find out, and at present unproved risk, is that of great astigmatism from the larger flap requisite.

"6. The larger wound certainly makes iris prolapse more likely, if there is not an iridectomy performed as part of the operation."

SOME NEUROSES FROM REFRACTIVE ERRORS.

Austin O'Malley (*American Medicine,* April) points out the close connection of the nerves of the eye and its appendages with the nerves supplying other parts of the body, and states that "a group of nerve-cells disconnected from the general nervous system, as glands, muscles, adipose, mucous, or other tissues may be disconnected, is not possible." He reviews briefly the anastomoses of the second, third, fourth, fifth, sixth and seventh cranial nerves with each other and with the sympathetic and through it with the pneumogastric, and also with the great occipital nerve, through the sympathetic and the second cervical. Irritation of any one of these nerves may cause a reflex irritation of other nerves, either adjoining the original one or possibly far removed from it. Thus it comes to pass that the continued use of eyes in which there are errors of refraction, such as hypermetropia or astigmatism, the strain put upon the ciliary muscles causes irritation of the ciliary nerves and this irritation is conveyed to the fifth or sensory nerve, through the lenticular ganglion, and causes pain in those parts of the head supplied by the

ophthalmic and superior maxiliary divisions of the fifth nerve. Or the motor nerves of the eyes may be similarly irritated and such conditions as nystagmus, hippus, and chorea-like winking produced. Where the irritation is transmitted to the sympathetic vaso-motor nerves, congestion of the conjunctival vessels follows, and when it passes through the sympathetic to the vagus, nervous dyspepsia and nausea may be produced.

Since a large percentage of cases of headache are due to eye-strain the eyes should be carefully examined among the first things done in seeking for the cause. In neurasthenia it is important to correct any errors of refraction, and many cases of nervous dyspepsia, especially in women who use their eyes a great deal for near work, will not be cured until this source of irritation is removed.

While in northern Europe a patient may be entirely relieved by refracting him down to a half-dioptre of what he actually needs; in this country it is often necessary to refract as closely as to one-eighth of a dioptre.

TOXIC AMBLYOPIA WITH REPORT OF TWENTY-FOUR CASES.

L. Stricker (*Lancet-Clinic,* May 2nd) reports twenty-four cases of toxic amblyopia, classified, as to the toxic agent producing the trouble, as follows: wood alcohol, 2; chronic ethyl, or grain alcohol, 4; chronic tobacco, 2; chronic alcohol and tobacco, 2; chronic alcohol and lead, 7; chronic lead, 5; gout, 5; and carbon bisulphid, 1. He mentions quinin, felix mass, arsenic, iodids, sulphids and chlorids as being other causes of amblyopia, but has had no experience with these. A careful study of his cases leads him to believe that in toxic amblyopia, the ganglion cells of the retina are diseased, primarily. In the acute form of the disease the intoxication is severe enough to cause the destruction of the ganglion cells very quickly, the poison acting directly and with considerably force. In the chronic form there is an impairment of the nutrition of the ganglion cells and the axis cylinders die first. In a mild form the poison may act directly upon the ganglion cells or their axis cylinders, or indirectly by its action on the vascular system, producing an arteriosclerosis, thus impairing the nutrition of both the retina and the optic nerve.

TOXIC AMBLYOPIA FROM WOOD ALCOHOL.

W. M. Carhart (*American Medicine,* April) reports a case of pronounced neuro-retinitis, followed by optic atrophy, caused by the inhalation of the fumes of wood alcohol present in shellac which he was applying to the interior of beer vats. After being engaged at this work for nearly three weeks, he was attacked with vertigo and nausea one day which compelled him to leave his work. The next day he returned to his work and in the afternoon his vision became greatly impaired and the nausea returned, followed by vomiting. By the following morning he was entirely blind in both eyes and ophthalmoscopic examination revealed marked swelling of both optic discs, practically obscuring them, and decided engorgement of the retinal vessels over the whole of the fundi.

The blindness persisted ten days in the left eye and two weeks in the right, in spite of vigorous treatment looking toward the elimination of the poison by diaphoresis and catharsis, followed later by the use of potassium iodid, and strychina hypodermatically. After this it gradually improved until it equalled 3/200 in the left eye and ability to see large objects with the right. There has been no improvement in the vision of either eye for some time, but the author thinks that he may retain practically what he has as the ophthalmoscopic appearance of the optic discs would indicate that there is still some circulation in the nerves. He emphasizes the importance of workmen, using shellac containing wood alcohol, taking the precautions of avoiding being confined in closed spaces any great length of time, and especially of leaving the work on the first symptom of trouble.

THE RADICAL CURE OF VERNAL CONJUNCTIVITIS.

L. Webster Fox (*Penn. Med. Jour.*, Dec., 1907), after referring to the distressing symptoms of vernal conjunctivitis and the long list of remedies that had been tried to alleviate these symptoms and cure the disease, all of which are apparently of little value, so far as a cure is concerned, describes his method of a radical cure by means of grattage, as follows:

"The upper eyelid is grasped along its margin by Darier forceps, and, the edge being turned upon itself, the lid is everted until the retrotarsal fold is brought into view. A horn spatula

should be inserted beneath the lid to protect the cornea. The exposed part is thoroughly scarified with a three-bladed scalpel. The granular tissue is then scrubbed with a toothbrush, which has been steeped in corrosive sublimate (1 to 500) solution just before being used. Immediately after scrubbing, the part is washed with a solution of the same strength. Another portion of the lid is now unrolled, and the scarifying and scrubbing and washing repeated until the whole conjunctival surface of the eyelid has been subjected to the treatment. If the lower lid is involved it should be treated the same way."

He has treated twelve cases in this way in ten of which the disease had been present not more than five years; the cure was permanent in these ten cases. In one case the operation had to be repeated and the time has been too brief to be certain of the ultimate result. In the other case the operation gave relief for some weeks after which the trouble returned. This was a very severe case and he is waiting an opportunity to repeat the operation at a time convenient to the patient.

ÆTIOLOGY AND TREATMENT OF CHRONIC CONJUNCTIVITIS.

Elschnig (*Deutsche Medizinische Wochenschrift*, June 25) calls attention to the chronic conjunctivitis due solely to excessive secretion of the Meibomian glands or to relative insufficiency of the eyelids, with defective closure during sleep. The chronic inflammation resulting from these causes can be readily cured; in the first case by squeezing out of the glands the excess of secretion, and in the second case by having the patients wear a linen band over the eyes, fastened to the ears, while they are in bed. The tarsal glands must be thoroughly evacuated every day by massage, pressing both lids together, the tarsal surfaces against each other, and the contents of the glands milked out with the fingers. This should be done every day at first and later as needed. In case of defective closure of the eyelids, the patient should push up the under lid occasionally during the day, and in severe cases a 3 per cent. boricized lanolin salve should be applied to the conjunctiva. Astringents are scarcely necessary, but compresses may prove useful.—*Jour. A. M. A.*

IMPERFORATION OF THE LACRIMONASAL DUCT IN THE NEW-BORN, AND ITS CLINICAL MANIFESTATIONS.

William Zeutmayer (*Jour. A. M. A.*, July 18) discusses the embryology, ætiology, complications, symptomatology, bacteriology, prognosis, diagnosis and treatment of this condition. Concerning the ætiology he says: "The possible causes of the condition manifesting itself as a discharge from the puncta, a distention of the sac or an abscess in the new-born, are sixfold. (1) Delayed separation and necrosis of the epithelial cells forming the cord from which the canal is formed. (2) Retention of the separated cells through imperforation of the septum between the lacrimonasal duct and the nasal chamber. (3) Through obstruction due to annular folds of the mucous membrane which may form at any point within the duct, Hüschke's and Hasner's valves. (4) Faulty development of the cartilages (D. Gunn). (5) Partial occlusion by pressure of inferior turbinate. (6) Stenosis from pressure exerted on the bones of the face during instrumental labor."

As to treatment, while many surgeons, including Weeks, Jackson, Peters and others, advise the use of some mild collyrium, solutions of silver salts, pressure and massage, giving nature time and opportunity to correct the trouble, the author agrees with others, such as Ollendorff, Koster, Cutler, Parsons and Mayou, who advise the use of probes and syringing. In his series of cases a cure followed the passing of a probe a single time in every case except one where it was necessary to probe three times before the case got well. He does not find it necessary to etherize the patient to pass the probe.

TO CLEAR THE CORNEA OF OPACITY FROM METALLIC CAUSTICS.

A. Guillery (*Deutsche Medizinische Wochenschrift*, June 25) finds it possible to clear up a recent opacity of the cornea produced by the action of lime, or other metallic caustics by the application to the cocainized eye of a mixture of ammonium chlorid and tartaric acid. He first uses a 4 per cent. solution of ammonium chlorid mixed with a solution of tartaric acid .02 to 1 per cent. in strength. Later the ammonium solution may be increased in strength to 10 per cent., but the strength of the solution of tartaric acid must not be increased.

DIFFUSE INTERSTITIAL KERATITIS IN ACQUIRED SYPHILIS.

A. E. Davis (*Jour. A. M. A.,* July 25) reports two cases of diffuse interstitial keratitis in patients who had no evidences of hereditary syphilis but who had contracted syphilis in early adult life. He reviews the literature on the subject which is rather meager, there having been comparatively few cases reported, discusses the pathology and differential diagnosis, and offers the following conclusions as the result of his study of the subject:

1. Diffuse interstitial keratitis may occur as a result of acquired syphilis.

2. It usually occurs as a late secondary sign of the disease, or during relapses in the tertiary stage of the general disease. Stephenson gives the average time of development of interstitial keratitis as 10.8 years after the primary sore. Loewinson has reported one case as early as three weeks after the appearance of a primary sore, while Ellett reports a case appearing as late as twenty-three years after the infection.

3. It almost invariably affects but one eye, although there are a few exceptions reported where both eyes were affected.

4. It runs a quicker and lighter course, as a rule, than the cases due to inherited syphilis, and is rarely harmful to the sight. It should be remembered, however, that Griffith has reported one case in which the sight was entirely lost.

5. True salmon-patches occur but seldom in these cases.

6. It is difficult to make a clinical diagnosis between the syphilitic and the tuberculous forms of the disease, and even a differential pathologic diagnosis is not always conclusive.

7. The prognosis is favorable, though it should be somewhat guarded from the fact that sight has been lost entirely in one case.